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CASE REPORT

Tourniquet-induced rhabdomyolysis after total knee replacement

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A case is described of a patient who developed rhabdomyolysis and acute renal failure after the use of an intraoperative tourniquet for elective orthopaedic surgery. A review of the literature revealed four similar cases in the last 20 years. The clinical features and management of such patients are discussed.

The use of tourniquets in orthopaedic surgery is common. Acute rhabdomyolysis is a well-recognised phenomenon in many fields of medicine, but the association with tourniquets is not well recognised.

Case report

A 73-year-old man with severe osteoarthritis of the hips and knees was admitted for a right-sided total knee replacement. His past medical history included chronic obstructive airways disease, angina, hypertension, cardiac failure and a gastric ulcer. On examination he was found to be plethoric, obese and dyspnoeic at rest. The examination was otherwise normal, apart from a widespread expiratory wheeze in his chest.

Preoperative chest radiograph, electrocardiogram, full blood count and serum biochemistry were unremarkable.

A standard right total knee replacement was performed by a consultant surgeon under a spinal anaesthetic. A well-padded mid-thigh pneumatic tourniquet was inflated to 350 mmHg for 92 min and the total anaesthetic time was 2 h.

In the immediate postoperative period, the patient became oliguric and was found to be pyrexial (39.1°C). The urine was noted to be cloudy and a serum biochemistry profile revealed a raised urea (10.4 mmol/l) and creatinine (298 mmol/l).

He was managed with intravenous fluids and broad-spectrum intravenous antibiotics, but despite adequate fluid replacement with central venous pressure monitoring his renal function continued to deteriorate (urea 20.2 mmol/l and creatinine 501 mmol/l).

On the 2nd postoperative day the tourniquet site was noted to be slightly swollen and tender when compared with the other side. A spot urine test was positive to myoglobin and the serum creatine kinase was very high at 16 000 IU (normal = < 270 IU). The clinical picture was thus in keeping with acute renal failure secondary to rhabdomyolysis and was treated with high-dose loop diuretic therapy and renal dose dopamine.

The urine output began to improve on day 4 with a corresponding reversal in the serum urea and creatinine.

On day 5 myoglobin was not detectable in the urine and the serum creatine kinase showed a downward trend.

One week later the renal function was back to normal and the patient began to mobilise. He was discharged on day 22 and was doing well on review in the outpatient department at 2 months.

Discussion

Tourniquets have been used for many years in surgery to obtain a 'bloodless field' and allow accurate dissection of the affected extremity. Esmarch first introduced the flat rubber bandage tourniquet in 1873, but there was a high incidence of associated peripheral nerve damage. Their use was superseded by the pneumatic tourniquet introduced by Harvey Cushing in 1904 *(1)*. However, the modern-day tourniquet is not free of complications, especially the damage of nerves beneath the tourniquet site *(2–7)*.

Rhabdomyolysis (the destruction of skeletal muscle *(8)*)

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and associated acute renal failure was first described by Waters in 1941 after crush injuries in the Battle of Britain. Rhabdomyolysis itself has been associated with many conditions since then, such as abnormal muscle states (eg polymyositis), drugs, infection, hyperthermia and various metabolic disturbances *(9)*.

Four other cases of torniquet-induced rhabdomyolysis have been described in the literature *(9–12)*. In these cases the torniquet time varied from 45 min to 4.5 h, and although renal function was preserved ultimately in all, two patients required a period of renal dialysis.

Many of the clinical features associated with acute rhabdomyolysis are non-specific, but pyrexia, pain and tenderness with oedema and haemorrhagic discoloration at the torniquet site, in association with oliguria, should alert the physician to the diagnosis. The diagnosis can then be confirmed by a spot urine or serum myoglobin level.

Other useful pointers are the presence of cloudy urine and significant elevations in serum creatine phosphokinase, lactate dehydrogenase and aspartate transaminase. The serum creatinine, urea, urate, phosphate and potassium also become increased, whereas the serum sodium and calcium concentrations become decreased *(8)*.

The optimum treatment is controversial and should be undertaken with the help of an experienced renal physician. Early recognition of the condition is vital. Aggressive fluid replacement with saline is essential in the early stages. Hyperkalaemia should be treated with insulin and glucose and intravenous calcium gluconate. After adequate fluid replacement intravenous mannitol or frusemide can be given to mitigate acute renal failure. Renal-dose dopamine may also be considered.

Urinary alkalinisation can be undertaken with sodium bicarbonate or lactate and hyperphosphataemia can be treated with phosphate binding antacids. Obviously, if these methods are not successful then both haemodialysis and peritoneal dialysis may be used *(8)*.

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